

REMARKS

This amendment is being filed in response to the Office Action mailed January 21, 2004. Claims 1 to 12 and 36 to 47 are under consideration.

Regarding the Amendments

The amendments to the claims are supported by the specification. In particular, the amendment to claims 1, 2, 8 to 12, 37 and 43 to 47 to recite "eosinophil" is supported, for example, at page 14, line 9, which discloses that galectin-3 may be a chemoattractant for eosinophils. The amendment to claim 1 to recite that the cell is "capable of having migration modulated by galectin-3" is supported, for example, at page 13, lines 23-25, which discloses that the cell type modulated may be any cell type that expresses galectin-3 and for which galectin-3 has an effect upon cell migration. The amendment to claim 12 to recite "decreasing" migration to a site of infection is supported, for example, at page 16, lines 16-18. The amendment to claims 36, 37 and 43 to 47, to recite a galectin-3 binding "antibody" is supported, for example, by claim 6, as originally filed. The amendment to claim 41, which depends from claim 36 or 37, was necessitated by the foregoing amendment to claims 36, 37 and 43 to 47. Thus, as the amendments to the claims are supported by the specification, no new matter has been added and entry thereof is respectfully requested.

I. REJECTIONS UNDER 35 U.S.C. §112

Written Description

The rejection of claims 1 to 12 and 36 to 47 under 35 U.S.C. §112, first paragraph, as allegedly lacking an adequate written description is respectfully traversed. The Examiner states that because "Applicants' invention is not limited to making changes in these known regions" and, allegedly, having "not adequately described any other regions which could be altered to modulate cell migration," an adequate written description of "modified galectin-3" or "galectin-3 fragments" allegedly has not been provided. [Office Action at pages 2, A., and 3, A.]

The claims prior to and following entry of the amendments are adequately described. Nevertheless, solely in order to further prosecution of the subject application and without

acquiescing to the propriety of the rejection, the claims have been amended as set forth above. The rejections will therefore be addressed as they may pertain to the amended claims.

Applicants first respectfully point out that there is no requirement that language reciting regions of a protein that can be altered be included in the claims to satisfy the written description requirement of 35 U.S.C. §112, first paragraph. In this regard, 35 U.S.C. §112, first paragraph reads in relevant part: “The specification shall contain a written description of the invention, and the manner and process of making and using it....” Emphasis added. Consequently, the written description can be found in the specification.

To satisfy the written description requirement, an applicant “must also convey with reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession of the invention.” *Vas-Cath, Inc. v. Mahurkar*, 935 F.2d 1555, 1563-64 (Fed. Cir. 1991). Possession may be shown by “any description of sufficient, relevant, identifying characteristics so long as a person skilled in the art would recognize that the inventor had possession of the claimed invention.” *Purdue Pharma L.P. v. Faulding Inc.*, 230 F.3d 1320, 1323 (Fed. Cir. 2000). The applicant, however, “does not have to describe exactly the subject matter claimed.” *In re Gosteli*, 872 F.2d 1008, 1012 (Fed. Cir. 1989)

A description of a genus may be achieved by a representative number of species falling within the scope of the genus or of a recitation of structural features common to the members of the genus, which constitute a substantial portion of the genus. *Reagents of the Univ. Calif. v. Eli Lilly* 119 F.3d 1559, 1568 (Fed. Cir. 1997), Emphasis added. For biological molecules, identifying characteristics can include, *inter alia*, sequence, structure and length. Although courts have not specified a minimum number of species constituting a representative number, or structural features common to the genus, an adequate written description does not require the disclosure of every species encompassed by the claims, even in an unpredictable art. *In re Angstadt*, 537 F.2d 498, 502-503 (CCPA 1976), Emphasis added. In *Lilly*, the court reiterated that “every species in a genus need not be described in order that a genus meet the written description requirement.” *Id. (citing Utter v. Hiraga*, 845 F.2d 993, 998-99 (Fed. Cir. 1988)). Thus, a description of every modified galectin-3 or every galectin-3 subsequence is clearly not required in order to satisfy the written description requirement of 35 U.S.C. §112, first paragraph.

Here, in view of the guidance in the specification and knowledge in the art at the time of the invention, numerous structural features of galectin-3 sequences would be known to the skilled artisan. Thus, the skilled artisan would be apprised of sufficient relevant identifying characteristics of a galectin-3 genus. In addition, the skilled artisan would know of numerous galectin-3 sequences having migration modulating activity and, therefore, a representative number of galectin-3 sequences.

Furthermore, because the skilled artisan would know galectin-3 sequence regions that confer particular function, and would also know conserved and non-conserved amino acids based upon species differences in galectin-3 sequences, the skilled artisan could predict amino acids that could be substituted or deleted without destroying cell migration modulating activity. As such, the skilled artisan would know of numerous modified sequences and fragments of galectin-3 capable of modulating cell migration. Consequently, the skilled artisan would know of a representative number of modified galectin-3 sequences and galectin-3 subsequences that modulate cell migration.

As set forth in the record, structural features of galectin-3 were known in the art at the time of the invention. As evidence of this knowledge, Exhibits A-C (Barondes *et al.*, J. Biol. Chem. 269:20807 (1994); Robertson *et al.*, Biochem. 29:8093 (1990); and Cherayil *et al.*, Proc. Natl. Acad. Sci. USA 87:7324 (1990), respectively) were previously submitted. Exhibits A and C each describe the location of various functional domains, including, *inter alia*, the carbohydrate (CHO) binding domain (lectin domain) located in C-terminal region of galectin-3 which mediates binding to receptor. Exhibit A indicates that the N-terminal half of galectin-3 mediates multimerization. Exhibit B indicates a sequence comparison among mammalian galectin-3, illustrating conserved and non-conserved regions. The specification discloses that both N-terminal and C-terminal domains of galectin-3 appear to be involved in migration-modulating activity. A C-terminal galectin-3 fragment (112 to 250; see, Yang *et al.*, Proc. Natl. Acad. Sci. USA 93:6737 (1996)) inhibited monocyte migration induced by full-length galectin-3 (page 15, lines 10-13; and page 32, lines 7-16). Thus, in view of the guidance in the specification and knowledge in the art, one skilled in the art would know of structural features shared among the galectin-3 genus, as well as regions that participate in cell migration modulating activity.

In view of the fact that N-terminal and C-terminal domains are important for migration increasing activity, the skilled artisan would know that galectin-3 sequences that increase migration would retain multimerization and CHO binding. Consequently, altered forms of galectin-3 sequences that retain migration increasing activity would also be known to the skilled artisan. As a non-limiting example to illustrate this point and not to limit the claims in any way, to produce modified galectin-3 sequences that increase cell migration, the skilled artisan would know that one or a few point mutations that do not destroy multimerization or CHO binding could be introduced into galectin-3. Selecting target amino acids for substitution or deletion could be based, for example, upon sequence homology among mammalian galectin-3 sequences, and knowledge of the structural features of galectin-3. For example, because human galectin-3 modulates cell migration in mice (see Examples 8 and 9, and Figure 12), the skilled artisan would know that mouse galectin-3 sequence could have one or many corresponding human galectin-3 amino acid substitutions without destroying galectin-3 cell migration increasing activity. Galectin-3 is about 250 amino acids in length (see Exhibit A), and there is 84% homology between human and murine galectin-3 proteins (see Exhibit B), meaning there are about 40 amino acid differences between murine and human galectin-3. In view of this knowledge, the skilled artisan could predict a large number of modified galectin-3 sequences that would retain cell migration increasing activity. For example, the skilled artisan would know that mouse galectin-3 could have one, two, three, four, five, or more amino acid substitutions corresponding to human galectin-3. As such, the skilled artisan would know of a representative number of altered galectin-3 sequences having cell migration increasing activity.

As to galectin-3 sequences that inhibit cell migration, because a C-terminal galectin-3 fragment (112 to 250) inhibited monocyte migration induced by full-length galectin-3, the skilled artisan would know that galectin-3 fragments lacking multimerization function but retaining CHO binding can inhibit cell migration. Consequently, the skilled artisan would also know of numerous galectin-3 fragments containing C-terminal lectin domain and lacking N-terminal multimerization domain that inhibit cell migration. As a non-limiting example to illustrate this point and not to limit the claims in any way, approximately 111 amino acids comprise the N-terminal portion of the 250 amino acid human galectin-3 sequence. The skilled artisan would know that galectin-3 fragments that inhibit cell migration could have all or a portion of this sequence removed. For example, the skilled artisan would know that, analogous to galectin-3

fragment 112 to 250, galectin-3 fragments having amino acids 111 to 250, 110 to 250, 109 to 250, 113 to 250, 114 to 250, 115 to 250, and so on, would inhibit cell migration. Consequently, the skilled artisan could predict numerous galectin-3 subsequences having cell migration inhibiting activity. As such, the skilled artisan would know of a representative number galectin-3 subsequences that inhibit cell migration.

In view of the fact that the skilled artisan would know of numerous structural features that identify the galectin-3 genus, as well as sequence regions that participate in cell migration modulating activity, the skilled artisan would know a representative number of galectin-3 sequences. Furthermore, because the skilled artisan could predict a large number of altered galectin-3 sequences having migration modulating activity, the skilled artisan would also know of a representative number of altered galectin-3 sequences. As such, an adequate written description for a genus of galectin-3 sequences is provided.

As to galectin-3 binding antibodies, the specification discloses that B2C10 can inhibit galectin-3 mediated cell migration (page 30, lines 15-19). Previously submitted Exhibit 1 (Liu *et al.*, Biochemistry 35:6073 (1996)) reported seven monoclonal antibodies that bind galectin-3. At least four of these antibodies, namely A3A12, B1A7, B3A12 and C1C2, activated galectin-3, as determined by potentiating galectin-3 binding to IgE and potentiating galectin-3 hemagglutination activity (Exhibit 1, Table 1). A3A12 also activated galectin-3, as determined by enhanced superoxide (SO) production of neutrophils. Thus, at least one antibody that inhibits cell migration and at least four antibodies that stimulate galectin-3 activity were known at the time of the invention.

In the previously submitted Declaration under 37 C.F.R. §1.132 (Exhibit 2), Dr. Liu concludes that based upon the studies described in Exhibit 1 and his expertise in the relevant art, at least one of the galectin-3 binding antibodies described in Exhibit 1 is expected to stimulate cell migration. This conclusion is a conservative estimate of the number of antibodies expected to modulate cell migration because in actuality four of the seven galectin-3 binding antibodies, namely A3A12, B1A7, B3A12 and C1C2, stimulated galectin-3 activity. Consequently, all of A3A12, B1A7, B3A12 and C1C2 antibodies may modulate cell migration. Accordingly, in view of the galectin-3 binding antibodies that decrease and increase cell migration, a representative number of galectin-3 binding antibodies are provided.

In sum, in view of the fact that the skilled artisan would know relevant identifying characteristics of the genus of galectin-3 sequences as well as regions that participate in cell migration modulating activity and, therefore, would also know a representative number of galectin-3 sequences that modulate cell migration, and further in view of the fact that the skilled artisan could predict a large number of altered galectin-3 sequences that modulate cell migration, an adequate written description of a genus of galectin-3 sequences that modulate cell migration is provided. Moreover, in view of the galectin-3 binding antibody that inhibits cell migration and the galectin-3 binding antibodies that activate galectin-3 an adequate written description of galectin-3 binding antibodies is also provided. As such, the rejection of claims 1 to 12 and 36 to 47 under 35 U.S.C. §112, first paragraph, for alleged lack of adequate written description must properly be withdrawn.

As to the rejection of claims 12 to 12 and 36 to 47 due to different cell types, claim 1 recites that the cell “expresses a galectin-3 receptor,” and, as amended, a “cell capable of having migration modulated by galectin-3.” Thus, only cells that 1) express galectin-3 receptor and; 2) whose migration can be modulated by galectin-3, are encompassed by claim 1 and claims depending therefrom. Accordingly, inoperative embodiments are excluded from these claims (*i.e.*, cells that do not express galectin-3 receptor and are incapable of having migration modulated by galectin-3), and relevant identifying characteristics shared among the cells encompassed by these claims are provided. Furthermore, the specification discloses four different cell types, namely monocytes, macrophages, neutrophils and eosinophils, whose migration is modulated by galectin-3 or galectin-3 binding antibody.

As to claims 2, 9 to 12, 37 and 43 to 47, these claims are directed to monocytes, macrophages, neutrophils and eosinophils, each of whose migration can be modulated by galectin-3 or galectin-3 binding antibody. Consequently, this grounds for rejection is not applicable to these claims.

In sum, in view of the fact that relevant identifying characteristics of the recited cells are provided, and four specific examples of cells that express galectin-3 and whose migration can be modulated by galectin-3 or galectin-3 binding antibody are disclosed, one skilled in the art would be apprised of the recited cell types. As such, an adequate written description of the recited cells

is provided and the rejection of claims 1 to 12 and 36 to 47 under 35 U.S.C. §112, first paragraph, is improper and must be withdrawn.

As the rejection based upon how to modulate migration *in vivo*, for example, to a tumor or inflammatory site, the grounds for this rejection are improper. In this regard, the rejection implies that Applicants are required to provide an explanation for “how administering galectin-3 to a wound site would permit the migration of cells to that site” and for “how a sufficient concentration of galectin-3 could be administered in order to allow for the migration of cells to an intended site.” [Office Action, page 4] However, the law does not require Applicant to explain how or why an invention works. Furthermore, the law does not require Applicant to specify a dosage if it is known that one skilled in the art could obtain a dosage without undue experimentation. Here, determining dosages for modulating cell migration are well within the level of the skilled artisan in view of the guidance in the specification and knowledge in the art (see, for example, page 10, lines 4-12; and page 19, lines 16-21)

Notwithstanding the foregoing, in an effort to fully respond to the Office Action, Applicants respectfully point out that the specification discloses that cells are recruited towards galectin-3 injected into mice *in vivo* (Examples 8 and 9, and Figure 11). The specification also discloses that contacting an inflammatory or infection site with galectin-3 or galectin-3 binding polypeptide can increase cell migration to the site (see, for example, page 17, lines 14-20; page 18, lines 6-9). Thus, in order to modulate cell migration, for example, locally to a tumor or site of infection, one skilled in the art would know to administer galectin-3 or galectin-3 binding antibody at or near the tumor or infection site.

In addition, means for sustained-release delivery of galectin-3 or galectin-3 binding antibody are disclosed in the specification and were known in the art at the time of the invention. For example, the specification discloses that transdermal skin patches, articles of manufacture such as devices capable of controlled delivery, can be used to deliver galectin-3 or galectin-3 binding polypeptide (see, for example, page 24, lines 3-8; page 25, line 12, to page 26, line 8). Slow release formulations, such as matrigel, *inter alia*, were known in the art at the time of the invention.

Thus, given the guidance in the specification and knowledge in the art, one skilled in the art would know or could readily ascertain without undue experimentation delivery of galectin-3

in order to modulate cell migration *in vivo*. Consequently, an adequate written description for modulating cell migration is provided and the rejection of claims 1 to 12 and 36 to 47 under 35 U.S.C. §112, first paragraph, for alleged lack of adequate written description, must properly be withdrawn.

Enablement

The rejection of claims 1 to 12 and 36 to 47 under 35 U.S.C. §112, first paragraph, as allegedly lacking enablement is respectfully traversed. The Examiner acknowledges that the specification “discloses assays for identifying fragments and subsequences of galectin-3” however, allegedly, “Applicants have not provided sufficient examples of these fragments and subsequences.” [Office Action at pages 4-6, 3A and 3B.]

Claims 5, 6 and 40 are adequately enabled by the specification. The proper standard for enablement under 35 U.S.C. §112, is whether one skilled in the art could make and use the invention without undue experimentation. As noted previously, “a considerable amount of experimentation is permissible, if it is merely routine, or if the specification in question provides a reasonable amount of guidance with respect to the direction in which the experimentation should proceed.” *In re Wands* 858 F.2d 731, 737 (Fed. Cir. 1988)

Applicants appreciate that the Patent Office acknowledges that the specification discloses routine assays for identifying galectin-3 fragments/subsequences that modulate cell migration. Given this acknowledgement, regardless of the number of examples of galectin-3 subsequences, the fact is that undue experimentation would not be required to obtain such galectin-3 subsequences. As such, the rejection should properly be withdrawn.

Notwithstanding the foregoing, the skilled artisan would know of and could predict a large number of galectin-3 subsequences that modulate cell migration such that the invention could be practiced without undue experimentation. As discussed above, the specification discloses that N-terminal and C-terminal domains of galectin-3 are important for galectin-3 cell migration modulating activity, and a C-terminal galectin-3 fragment with a lectin domain inhibits galectin-3 induced cell migration. As also discussed above and in the record, domains conferring particular galectin-3 functions were also known in the art at the time of the invention. Thus, in view of the guidance in the specification and knowledge in the art, in order to produce additional galectin-3 fragments that inhibit cell migration the skilled artisan would know to delete or

destroy the multimerization domain and retain the lectin binding domain of galectin-3. Again, a non-limiting example illustrating this point and not limiting the claims is that the skilled artisan would expect that galectin-3 subsequences having amino acids 111 to 250, 110 to 250, 109 to 250, 113 to 250, 114 to 250, 115 to 250, and so on, would inhibit cell migration. Thus, contrary to the Office Action, in view of the guidance in the specification and knowledge in the art, the skilled artisan could predict a large number of galectin-3 subsequences that inhibit cell migration. Accordingly, as the skilled artisan would know a large number of specific galectin-3 subsequences that inhibit cell migration, a sufficient number of galectin-3 fragments having migration inhibiting activity could be readily produced without undue experimentation (*e.g.*, using recombinant techniques) to practice the claimed invention.

Similarly, because the skilled artisan would know the function and location of lectin and multimerization domain regions, to produce a galectin-3 subsequence that stimulates cell migration, the skilled artisan would know to delete amino acids outside of these regions in order to retain this activity. The skilled artisan would therefore also know specific galectin-3 subsequences that stimulate cell migration, and could readily produce such sequences using recombinant techniques without undue experimentation to practice the claimed invention.

It appears that the Examiner does not question that galectin-3 subsequences that stimulate or inhibit cell migration can be produced, as set forth above and in the record. Rather, the grounds for rejection are because "Applicants have still not defined the residues outside these regions (lectin and multimerization domains) which are critical to inhibit cell migration." First, even if for the sake of argument every region of galectin-3 had not been characterized in respect to modulating cell migration, for the reasons set forth above the skilled artisan could predict a large number of different galectin-3 subsequences that stimulate or inhibit cell migration without doing any experimentation. Second, the claims do not include inoperative galectin-3 subsequences. Thus, even if galectin-3 subsequences do not modulate cell migration, such subsequences are not included within the claimed methods. Third, Applicants respectfully point out that the scope of enablement need only bear a "reasonable correlation" to the scope of the claims. *In re Fisher*, 427 F.2d 833, 839 (CCPA 1970) Consequently, enablement under 35 U.S.C. §112, first paragraph does not require that every region of a given protein be exhaustively characterized so that the skilled artisan knows each and every possible subsequence of the protein that could be used in a claimed method.

Moreover, the fact is that if the skilled artisan wished to produce additional galectin-3 subsequences, the specification discloses routine assays for identifying galectin-3 sequences that modulate cell migration. Producing recombinant proteins was routine in the art at the time of the invention, and *in vitro* and *in vivo* cell migration assays also were routine (see, for example, page 9, lines 5-8; and page 28, line 3, to page 29, line 6). Thus, the skilled artisan would not have to “predict” galectin-3 subsequences having cell migration modulating activity. In this regard, the court recognized long ago that screening hybridoma cells to determine which produced monoclonal antibodies having a particular binding characteristic did not require undue experimentation. *In re Wands*, 858 F.2d 731 (Fed. Cir. 1988) Here, given that 1) producing recombinant proteins was routine; and 2) cell migration assays were routine, the skilled artisan could obtain additional galectin-3 subsequences that modulate cell migration without undue experimentation.

As to galectin-3 binding antibodies, as discussed above, B2C10 galectin-3 binding antibody inhibits cell migration and four antibodies, namely, A3A12, B1A7, B3A12 and C1C2 were known to stimulate galectin-3 activity. Consequently, specific examples of antibodies that modulate cell migration are disclosed.

In addition, the Examiner will appreciate that methods of producing antibody mutants, including humanized and fully human forms as well as antibody subsequences were routine in the art at the time of the invention (see, for example, page 15, line 16 to page 16, line 10). Given that regions conferring antibody structure and function were also known in the art at the time of the invention. Thus, the skilled artisan could modify any of B2C10, A3A12, B1A7, B3A12 and C1C2 to produce alternate antibody forms having galectin-3 inhibiting or activating activity without undue experimentation. Consequently, the skilled artisan could make a large number of galectin-3 binding antibodies that modulate cell migration without undue experimentation.

In sum, in view of the guidance in the specification and knowledge in the art, the skilled artisan could predict a large number of galectin-3 subsequences, as well as galectin-3 binding antibodies, that modulate cell migration. The skilled artisan, using routine methods disclosed in the specification or known in the art, could readily obtain additional galectin-3 subsequences as well as galectin-3 binding antibodies that modulate cell migration without undue experimentation. Thus, as galectin-3 subsequences as well as galectin-3 binding antibodies that

modulate cell migration could be obtained without undue experimentation, claims 1 to 12 and 36 to 47 are adequately enabled. As such, the rejection under 35 U.S.C. §112, first paragraph must properly be withdrawn.

The rejection of claims 1 to 12 and 36 to 47 under 35 U.S.C. §112, first paragraph, as allegedly lacking enablement is respectfully traversed. The Examiner acknowledges that the specification is enabling “for the use of galectin-3 to stimulate monocytes” but, allegedly, “does not reasonably provide enablement for....migration of cells other than monocytes.” In addition, the specification allegedly does not adequately enable “methods to treat the claimed conditions, such as inflammation, infection or cancer.” [Office Action at pages 6-7, 3C.]

As set forth above, the amended claims require that the cell “expresses a galectin-3 receptor,” and be “capable of having migration modulated by galectin-3.” Thus, only cells that express galectin-3 receptor and whose migration can be modulated by galectin-3 are encompassed by claim 1 and claims depending therefrom. Accordingly, inoperative cell type embodiments are excluded from the claims.

As to the assertion that only “monocyte” migration can be modulated with galectin-3 or a galectin-3 binding antibody, Applicants respectfully disagree. In this regard, the specification discloses that neutrophil migration can be modulated (see, for example, page 35, lines 11-15; and Figure 11). The specification also discloses that macrophage migration can be modulated (see, for example, page 34, line 24, to page 35, line 6; and page 35, line 24, to page 36, line 2). Eosinophil migration can also be modulated in accordance with the claimed methods (see, for example, Figure 11; and, corroborated in Sano *et al.*, *J. Immunol.* 165:2156 (2000)). Thus, the specification discloses that migration of all four of these cell types can be modulated with galectin-3 or galectin-3 binding antibody.

As to “predicting” additional cell types whose migration could be modulated with galectin-3 or a galectin-3 binding antibody, the skilled artisan need not make any prediction but, rather, would merely screen cells for migration using the routine assays disclosed in the specification or known in the art. Thus, given that cell migration assays are routine, the skilled artisan could identify additional cell types whose migration can be modulated by galectin-3 or galectin-3 binding antibody without undue experimentation.

Finally, as to enabling “methods to treat the claimed conditions, such as inflammation, infection or cancer,” claims 1 to 12 and 36 to 47 do not recite treating inflammation, infection or cancer. Consequently, treating these conditions need not be enabled in order to enable claims 1 to 12 and 36 to 47. As such, this ground for rejection is improper and must be withdrawn.

In sum, in view of the fact that the specification discloses that migration of four different cells types can be modulated by galectin-3 or galectin-3 binding antibody, that cell types whose migration is not modulated by galectin-3 or galectin-3 binding antibody are excluded from the claims, and that the skilled artisan could readily identify other cells whose migration is modulated by galectin-3 or galectin-3 binding antibody, undue experimentation would not be required to practice the invention with additional cell types. Further in view of the fact that the claims do not recite treating inflammation, infection or cancer, such treatment methods need not be enabled. As such, claims 1 to 12 and 36 to 47 are adequately enabled and the rejection under 35 U.S.C. §112, first paragraph, for alleged lack of enablement must properly be withdrawn.

CONCLUSION

In summary, for the reasons set forth herein, Applicants maintain that claims 1 to 12 and 36 to 47 clearly and patentably define the invention, respectfully request that the Examiner reconsider the various grounds set forth in the Office Action, and respectfully request the allowance of the claims which are now pending.

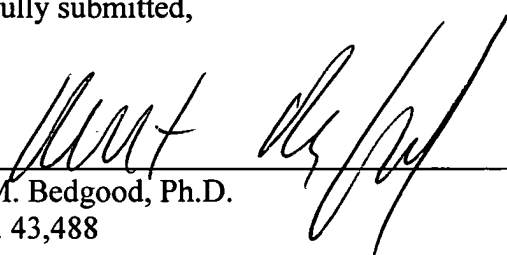
If the Examiner would like to discuss any of the issues raised in the Office Action, Applicant's representative can be reached at (858) 509-4065.

Please charge any additional fees, or make any credits, to Deposit Account No. 03-3975.

Respectfully submitted,

Date: _____

7.20.04



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